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Health Effects Assessment for Acenaphthylene

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16. ABSTRACT

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with specific chemicals or compounds. The Office of Emergency and Remedial Response (Superfund) uses these documents in preparing cost-benefit analyses under Executive Order 12991 for decision-making under CERCLA. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data are available. The interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed. Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, RfD_S or subchronic reference dose, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval. The RfD is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan. For compounds for which there is sufficient evidence of carcinogenicity, q_1^*s have been computed, if appropriate, based on oral and inhalation data if available.

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PREFACE

This report summarizes and evaluates information relevant to a preliminary interim assessment of adverse health effects associated with acenaphthylene. All estimates of acceptable intakes and carcinogenic potency presented in this document should be considered as preliminary and reflect limited resources allocated to this project. Pertinent toxicologic and environmental data were located through on-line literature searches of the TOXLINE, CANCERLINE and the CHEMFATE/DATALOG data bases. The basic literature searched supporting this document is current up to May, 1986. Secondary sources of information have also been relied upon in the preparation of this report and represent large-scale health assessment efforts that entail extensive peer and Agency review. The following Office of Health and Environmental Assessment (OHEA) sources have been extensively utilized:

U.S. EPA. 1980a. Ambient Water Quality Criteria Document for Polynuclear Aromatic Hydrocarbons. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Water Regulations and Standards, Washington, DC. EPA 440/5-80-069. NTIS PB 81-117806.

U.S. EPA. 1980b. Hazard Profile for Acenaphthylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Solid Waste, Washington, DC.

U.S. EPA. 1983. Reportable Quantity Document for Acenaphthylene. Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Emergency and Remedial Response, Washington, DC.

The intent in these assessments is to suggest acceptable exposure levels whenever sufficient data were available. Values were not derived or larger uncertainty factors were employed when the variable data were limited in scope tending to generate conservative (i.e., protective) estimates. Nevertheless, the interim values presented reflect the relative degree of hazard associated with exposure or risk to the chemical(s) addressed.

Whenever possible, two categories of values have been estimated for systemic toxicants (toxicants for which cancer is not the endpoint of concern). The first, RfD_S (formerly AIS) or subchronic reference dose, is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs during a limited time interval (i.e., for an interval that does not constitute a significant portion of the lifespan). This type of exposure estimate has not been extensively used, or rigorously defined, as previous risk assessment efforts have been primarily directed towards exposures from toxicants in ambient air or water where lifetime exposure is assumed. Animal data used for RfD_S estimates generally include exposures with durations of 30-90 days. Subchronic human data are rarely available. Reported exposures are usually from chronic occupational exposure situations or from reports of acute accidental exposure. These values are developed for both inhalation (RfD_{SI}) and oral (RfD_{SO}) exposures.

The RfD (formerly AIC) is similar in concept and addresses chronic exposure. It is an estimate of an exposure level that would not be expected to cause adverse effects when exposure occurs for a significant portion of the lifespan [see U.S. EPA (1980a) for a discussion of this concept]. The RfD is route-specific and estimates acceptable exposure for either oral (RfD_O) or inhalation (RfD_I) with the implicit assumption that exposure by other routes is insignificant.

Composite scores (CSs) for noncarcinogens have also been calculated where data permitted. These values are used for ranking reportable quantities and the methodology for their development is explained in U.S. EPA (1983).

For compounds for which there is sufficient evidence of carcinogenicity RfD_S and RfD values are not derived. For a discussion of risk assessment methodology for carcinogens refer to U.S. EPA (1980a). Since cancer is a process that is not characterized by a threshold, <u>any</u> exposure contributes an increment of risk. For carcinogens, q_1^*s have been computed, if appropriate, based on oral and inhalation data if available.

ABSTRACT

Data sufficient for risk assessment were not available for acenaphthylene; therefore, no risk assessment values were derived. A Russian inhalation study where rats were exposed to acenaphthylene dust suggests a carcinogenic role for this compound (Rotenberg and Mashbits, 1965). The lungs appear to be a target organ for acenaphthylene by either inhalation (Rotenberg and Mashbits, 1965; Reshetiuk et al., 1970) or oral (Rotenberg and Mashbits, 1965) exposure. Continued mutagenicity testing and short-term animal carcinogenicity assays to clarify the carcinogenic role of acenaphthylene are recommended as a first step in the testing of acenaphthylene.

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LIST OF ABBREVIATIONS

CAS	Chemical Abstract Service
CS	Composite score
K_{ow}	Octanol/water partition coefficient
LD_{50}	Dose lethal to 50% of recipients
MED	Minimum effective dose
NOEL	No-observed-effect level
PAH	Polycyclic aromatic hydrocarbon
ppm	Parts per million
RV_d	Dose-rating value
RV_e	Effect-rating value

1. ENVIRONMENTAL CHEMISTRY AND FATE

Selected physical and chemical properties and environmental fate of acenaphthylene are presented in Table 1-1.

Acenaphthylene is widely distributed in the environment. In air, acenaphthylene will be present partly in the vapor phase and partly in the sorbed state onto aerosol particles. Acenaphthylene adsorbed to particulates could potentially travel great distances before ultimately being removed by rainfall or dry deposition (NRC, 1983; Pankow et al., 1984). Acenaphthylene in the vapor phase is expected to undergo direct photolysis or oxidation by reaction with hydroxyl radicals or ozone (estimated oxidation half-life ~1 hour) (HSDB, 1986; NRC, 1983; U.S. EPA, 1986a). In water, acenaphthylene will be present partly in solution and partly as sorbed onto suspended particles and sediments. The dissolved portion may undergo rapid hydrolysis and significant biodegradation (Callahan et al., 1979). The adsorbed portion may persist for years in sediments (Bjoerseth et al., 1979). Based on the relatively high log K_{ow} value, acenaphthylene is expected to strongly adsorb to soil, thus persisting in the upper few centimeters. In soil, it may undergo biodegradation.

Human exposure data specific for acenaphthylene could not be located in the available literature. U.S. EPA (1980a), however, reviewed monitoring studies in which total PAH content of groundwater varied from 0.003-0.04 µg/ℓ and total PAH content of surface water ranged from 0.24-2.5 µg/ℓ. In finished drinking water from 15 U.S. cities, total PAH content ranged from 0.3-138.5 ng/ℓ, with samples from only two cities >10 ng/ℓ (Basu and Saxena, 1977; 1978). The other major sources of oral exposure of humans to PAH is through food. Borneff (1977) estimated this contribution

TABLE 1-1

Selected Physical and Chemical Properties
and Environmental Fate of Acenaphthylene

CAS number:	208-96-8	
Chemical class:	polycyclic aromatic hydrocarbon	
Molecular weight:	152.20	
Vapor pressure:	10^{-3} to 10^{-2} mm Hg at 20°C (estimated)	Callahan et al., 1979
Water solubility:	3.93 mg/ℓ at 25°C (estimated)	Callahan et al., 1979
Log octanol/water partition coefficient:	4.07 (estimated)	Callahan et al., 1979
Bioconcentration factor:	18.0 green mussels (Perna veridis)	Hungspreugs et al., 1984
Half-lives in		
Air:	NA	
Water:	NA	
Soil:	NA	

NA = Not available

at ~3-4 mg total PAH/year. These data do not indicate what proportion of total PAH content of water and food is acenaphthylene, so it is not possible to estimate human intakes of acenaphthylene alone. The data reviewed and presented by U.S. EPA (1980a) imply that acenaphthylene may be a small proportion of the total PAH content of water and food, since this compound is not listed by name in tables that present levels of the more abundant PAHs.

Overall, human exposure to acenaphthylene may be expected to occur by both oral and inhalation routes and it is impossible to predict which route may be the more important.

2. ABSORPTION FACTORS IN HUMANS AND EXPERIMENTAL ANIMALS

Pertinent data regarding the absorption of acenaphthylene following oral or inhalation exposure could not be located in the available literature.

3. TOXICITY IN HUMANS AND EXPERIMENTAL ANIMALS

3.1. SUBCHRONIC

3.1.1. Oral. In a Polish study reported in an abstract, Knobloch et al. (1969) administered acenaphthylene orally to rats at a dose of 0.6 g/kg for 40 days. Treatment-related effects observed were considerable body weight loss, changes in renal function, changes in the peripheral blood pattern and increased serum aminotransferase activities.

In a Russian study (Rotenberg and Mashbits, 1965), acenaphthylene in oil was administered orally to white mice at a dose 1/10 the LD_{50} [LD_{50} = 1760 (range of 2800 to 1100) mg/kg] every other day for 2 months. Treated mice showed a significant lag in weight gain as compared with controls. Histopathological examination of organs showed signs of stasis in the parenchymatous organs and albuminoid degeneration of the liver. The most severe changes were observed in the lungs, which showed hemorrhage with destruction of the interalveolar septa and focal bronchial pneumonia. Purulent foci were observed in isolated cases, and bronchogenic lung cancer was diagnosed in one mouse. Further details of this study were not provided.

3.1.2. Inhalation. In a Russian study (Rotenberg and Mashbits, 1965), acenaphthylene was administered to white rats intratracheally in a sunflower oil solution or by blowing acenaphthylene powder into the trachea. The dosing regimen used was not provided. The pulmonary tracts of animals sacrificed 1 month after the experiment began showed signs of tracheobronchitis and hyperemia, edema and necrosis of the epithelium in the trachea and bronchi with the formation of ulcers. No further details of this study were available.

In an additional study reported by Rotenberg and Mashbits (1965), white rats were exposed to acenaphthylene dust at 0.5-1.25 mg/m³ for 4 hours/day for 4 months. After 3 weeks of exposure, a delay in weight gain and a tendency toward decreased blood pressure were observed. Histopathological examination revealed various degrees of malignancy in the lungs of almost all treated rats. Focal bronchitis and peribronchitis with bronchiolization of the alveolar and metaplasia of the bronchial epithelium were observed in the mildest cases. Advanced cases showed desquamation of the bronchial and alveolar epithelium, papillar growths in the epithelium and, in three rats, isolated regions of carcinoma in the form of strands of epithelial cells. Further details of this study were not provided.

In a Russian study by Reshetiuk et al. (1970), ~100 white male rats were exposed to vapors of acenaphthylene at a concentration of 18±2.5 mg/m³, 4 hours/day, 6 exposures/week for 5 months. In exposed rats, reflexes of the upper airways were altered and an increase in the concentration of nucleic acids in the liver was observed. Histopathological examination of the lungs revealed aspecific pneumonia as the major pathology of inhalation exposure to acenaphthylene. Changes observed in the lungs included desquamation of the cells in the alveolar epithelium and focal bronchitis accompanied by hyperplasia and metaplasia of the bronchial epithelium No signs of malignant growth were observed in this study. No further details of this study were available.

3.2. CHRONIC

Pertinent data regarding the toxic effects of acenaphthylene following chronic oral or inhalation exposure could not be located in the available literature.

3.3. TERATOGENICITY AND OTHER REPRODUCTIVE EFFECTS

Pertinent data regarding teratogenic and other reproductive effects following oral or inhalation exposure could not be located in the available literature.

3.4. TOXICANT INTERACTIONS

Without providing details, Reshetiuk et al. (1970) stated that inhalation of naphthalene caused an increase in the sensitivity of rats to a single inhalation exposure of acenaphthylene at 7 mg/m³. Observations recorded include an increase in oxygen "composition" (as written by translator -- probably should read "consumption") (60%), an increase in body (0.6±0.2°) and skin temperature (0.4±0.2°), an increase in blood peroxidase activity (38%), and decreases in blood sugar concentration (10%) and ascorbic acid in the lungs (33%).

4. CARCINOGENICITY

4.1. HUMAN DATA

Pertinent data regarding the carcinogenic potential of acenaphthylene in humans following oral or inhalation exposure could not be located in the available literature.

4.2. BIOASSAYS

4.2.1. Oral. Pertinent data regarding the oncogenicity of acenaphthylene in orally exposed animals could not be located in the available literature.

4.2.2. Inhalation. Rotenberg and Mashbits (1965) reported "various degrees of malignancy" in the lungs of almost all of an unspecified number of rats exposed to acenaphthylene dust at 0.5-1.25 mg/m³ for 4 hours/day for 4 months. Further details were not provided. In another Russian study with male white rats, histopathological lesions including hyperplasia and metaplasia of the bronchial epithelium, but no signs of malignancy, were reported following inhalation of acenaphthylene vapors at 18 mg/m³, 4 hours/day, 6 days/week for 5 months (Reshetiuk et al., 1970).

4.3. OTHER RELEVANT DATA

Acenaphthylene has tested negative for reverse mutations in Salmonella typhimurium strains TA1537 and TA1538 with S-9 metabolic activation (Gatehouse, 1980) and in strains TA98 and TA100 with and without S-9 from 3-methylcholantrene induced rats (Florin et al., 1980). Acenaphthylene was mutagenic in S. typhimurium strain TM677 with S-9 metabolic activation (Kaden et al., 1979).

4.4. WEIGHT OF EVIDENCE

Acenaphthylene has not been evaluated for its carcinogenicity in humans and has not been adequately studied for its carcinogenic activity in

animals; therefore, it can be placed in IARC Group 3 and in EPA Group D, not classified, according to the CAG classification scheme (U.S. EPA, 1986b). Given its structural realtionship to known carcinogenic PAHs, however, prudent public health policy would dictate minimizing exposure until more is known about the health hazards.

5. REGULATORY STANDARDS AND CRITERIA

No standards or criteria are available for acenaphthylene alone. The U.S. EPA (1980a) derived ambient water quality criteria for PAH based upon the excess cancer risk associated with benzo(a)pyrene, a known animal carcinogen. For excess cancer risks of 10^{-5}, 10^{-6} and 10^{-7}, these criteria are 28.0, 2.8 and 0.28 ng/ℓ, respectively, for consumption of 6.5 g of aquatic organism and 2 ℓ of water/day. Based on consumption of aquatic organisms alone, these criteria are 311.0, 31.1 and 3.11 ng/ℓ, respectively.

6. RECOMMENDATIONS

Because of the lack of data for the carcinogenicity and threshold toxicity of acenaphthylene, risk assessment values cannot be derived.

Acenaphthylene is a PAH, a class of chemicals that includes known carcinogens. In a Russian study, malignancies in the lungs of rats were associated with inhalation exposure to acenaphthylene dusts (Rotenberg and Mashbits, 1965). Carcinogenicity testing of acenaphthylene should be a priority. Mutagenicity studies of acenaphthylene have been negative in S. typhimurium strains TA1537, TA1538, TA98 and TA100 (Gatehouse, 1980; Florin et al., 1980), but positive in strain TM677 (Kaden et al., 1978).

If adequate testing indicates that acenaphthylene is not carcinogenic, effort should be made to determine thresholds for noncarcinogenic toxicity. Exposure data specifically for acenaphthylene could not be located, but assuming exposure similar to other PAHs, both oral and inhalation routes of exposure may be of concern. From the Russian studies (Rohenberg and Mashbits, 1965; Reshetiuk et al., 1970), the lungs appear to be the target organ; additional studies may define this further. Continued mutagenicity testing, particularly in eukaryotic systems, and short-term animal assays may be useful to qualitatively estimate the oncogenicity of this compound.

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